as well as provide the increased power necessary to investigate differences in potential effect modifiers, such as asthma severity or school distance from a highway. Although our original intent in the study was to investigate differences between schools, this was not possible because of the lack of simultaneous school-site measurements and observed similarities in exposure levels among all schools. Because all schools were located in a dense urban locale, schools located farther away from highways were still affected by local traffic (see Spira-Cohen et al. 2010).

An additional limitation of this study was the lack of adequate data on daily medication use. Many of the children who reported some use of controller medications took them as needed, rather than on a regular basis, which we suspect may reflect findings of underuse of controller medications in populations similar to that of our study (i.e., blacks and Hispanics of lower socioeconomic status) (e.g., Finkelstein et al. 2002). Because the daily medication habits of the participants did not change before or during the study, the observed associations are not likely to be attributable to confounding by medication use.

Although our symptom score collection methodology may have the potential for reporting bias, we do not expect this bias to confound our results because the subjects were unaware of their personal pollution measurements. Similarly, all subjects had the same potential for measurement error from the backpack samplers, which were attached to the backpack as close as possible to the breathing zone (waist height or above).

A further limitation of this study is that our EC measurements could indicate other properties of traffic-related air pollution that are correlated with EC. We acknowledge that other pollutants that we did not measure in this study, such as ultrafine particle concentrations, kicked-up road dust, and/or particles from tire wear, cannot be ruled out as potential confounders of exposure.

Conclusion

A major strength of this study was the ability to obtain daily measures of personal exposure to EC (rather than using $PM_{2.5}$ mass, distance from roadway indices, or central-site data). Past traffic-related air pollution studies investigating health effects have relied mainly on central-site monitoring data, modeled exposure variables, or employed proximity to roadway as an exposure metric. In addition, this study found similar, albeit weaker, associations using school-site monitoring for EC, suggesting that school-site stationary measurements of EC may be representative of average daily personal exposures across the study participants in this dense urban setting. However, we found the strongest health–EC associations with the more accurate personal measure of "actual" exposure.

Using personal measurements, our findings more definitively confirm those of other recent urban exposure–asthma studies that have also pointed to the carbonaceous fraction of the PM, rather than total $PM_{2.5}$ mass, as showing stronger associations with adverse respiratory health. Therefore, exposure–health effects studies that rely on exposure measures of PM mass from central-site monitors may be underestimating health relationships with individual components of the PM.

Correction

In Results ("Symptom–PM analysis"), risks and 95% CIs were presented as percentages in the original text published online. They have been converted to RRs here.

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